Modern Concepts of Cardiovascular Disease

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NORMAL ELECTROCARDIOGRAMS IN THE PRESENCE OF CORONARY DISEASE

In a population like that of the United States, the average age of whose components is continuously increasing, methods to discover premature senility become popular. The electrocardiograph is a peculiarly appealing instrument as it is commonly supposed to possess a prescience in the discovery of coronary pathology superior to any other technique. The purpose of this paper is to emphasize some of its limitations in this direction and to question some terminology.

Recent techniques, such as those of Blumgart and Schlesinger, Robb, and Lowe, are approaching a better explanation of the circulatory balance of the coronary system, and are redefining the differences between "coronary thrombosis", and "cardiac infarction"— terms which are used loosely as equivalents by many practitioners. Even more obscure are the terms, "coronary sclerosis", "coronary narrowing", and "coronary disease", when used as refuges in electrocardiographic interpretation. As Blumgart and Schlesinger have shown, when coronary pathology reaches the degree necessary to produce angina pectoris, coronary occlusion has already occurred in one to many areas of the coronary system. It seems reasonable now to become accustomed to referring to coronary thrombosis much more frequently than before, and to confine our electrocardiographic interpretations of coronary pathology to degrees of myocardial infarction.

This leaves us, it is true, with the problem of transient myocardial ischemia with electrocardiographic changes, or, if you will, with a myocardial infarct which did not quite occur. In such a condition, it is fair, at the moment, to question whether small areas of acute infarction may not actually have been present which later were undiscovered by routine pathological technique. A similar reemphasis has ocurred in the vascular pathology of the brain. Episodes of transient paralysis, memory loss, aphasia, and the like have been referred to in the past as due to cerebro-vascular spasm. Doubtless such spasms in cerebral arteries can occur as shown by certain instances of transient blindness with demonstrable spasm of retinal vessels. However, more meticulous study of brain sections reveals that these cerebro-vascular "spasm" episodes are indeed minute thromboses, venous or arterial, with very localized areas of infarction.

Recently, another cardiac syndrome with an electrocardiographic pattern has been described, that of acute coronary insufficiency with myocardial infarction, to be distinguished from coronary occlusion with infarction. A discussion of this has been published by Master, Gubner, Dack and Jaffee. Stroud has also described cases of silent or atypical coronary occlusion without characteristic electrocardiograms. In the former syndrome, the pathological changes are mainly subendocardial, often involving the septal and papillary muscle regions as a patchy process not infarcting the whole thickness of the heart wall. Such a process is said to result in depression of S-T segments of the electrocardiogram rather than elevation, and it does not produce Q waves. Coronary insufficiency of this nature can be caused by factors increasing heart work, or decreasing coronary flow.

In all of this problem, however, the anatomical changes in the heart muscle must be considered in terms of dynamic circulatory balance. The rapidity of occlusion of a coronary vessel is the significant factor as mediated by the existing, or potential, collateral circulation, and the demands upon the heart during a period of ischemia.

We are much concerned at present with discovering the range of normal in the heart, and particularly in the electrocardiogram. Perhaps as important is discovering the range of abnormal hearts with normal, or at most, only suspicious electrocardiograms. An appreciation of the extent of this twilight zone should at least prevent dogmatism in electrocardiographic interpretation.

Much of our interest in the electrocardiogram in recent years has been centered upon the S-T and T segments. The S-T segment elevations and depressions, particularly at the junction with the QRS, are of most importance. T wave directions and amplitude are often diagnostic. On the other hand, it is not possible to make a diognosis of coronary disease from the electrocardiogram unless, (1) the findings are those of myocardial anoxemia of a critical degree (2) other causes of S-T and T segment changes are ruled out.

Without the hope of its being complete the following list of conditions, other than coronary atherosclerosis, responsible for abnormalities of S-T and T segments is submitted.

1—Drugs
- a. Digitalis
- b. Quinidine
- c. Tobacco

2—Myocardial Infection
- a. Rheumatism
- b. Diphtheria
- c. Trichinosis

3—Other General Infections
- a. Pneumonia

4—Pericarditis

5—Toxemia and Metabolic Disorders
- a. Uremia
- b. Diabetic acidosis
- c. Hypocalcemia
- d. Hyperthyroidism
- e. Hypothyroidism
- f. Insulin shock
- g. Addison's disease
- h. Anemia
- i. Avitaminosis—especially beri-beri
- j. Acidosis
- k. Alkalosis
- l. Anoxemia
- m. Shock

6—Changes in posture of the patient

7—Axis deviation

8—Abnormal heart rhythms
- a. Paroxysmal tachycardia and auricular fibrillation
- b. Sinus tachycardia

9—Alterations in vago-sympathetic tone
- a. Pain
- b. Fear
- c. Anesthesia

10—Pulmonary embolism

11—Miscellaneous
- a. Periarteritis nodosa
- b. Trauma
- c. Malignancy of myocardium
- d. Dissecting aneurysm of coronary artery
- e. Dissecting aneurysm of aorta
- f. Syphilis of aorta
- g. Gall bladder disease
- h. Scleroderma
- i. General Anasarca
- j. Cooling the heart through ingestion of ice water
- k. Terminal states of all kinds
- l. Unexplained

Such a list must be considered before an attempt can be made to diagnose coronary degeneration from the electrocardiogram. Similar, but less extensive lists could be compiled for auriculoventricular, or intraventricular block.

The converse of these observations concerns individuals with angina pectoris and occlusive processes in coronary arteries with normal electrocardiograms. Such electrocardiograms are not difficult to find since about twenty per cent of individuals with angina, when first seen, have an entirely negative examination including electrocardiogram and x-ray of the heart. A more convincing group, however, are those coming to autopsy with various grades of coronary pathology who have died, in many instances, of conditions unrelated to the heart. In such a group, wholesome surprises await us all at the autopsy table. These cases may be divided into six classifications:

1. Coronary sclerosis with or without calcification of high degree, but with wide lumen.
2. Coronary sclerosis with or without calcification but with narrowing of significant amount.
3. Coronary occlusion—complete.
4. Coronary occlusion with recanalization.
5. Coronary occlusion with myocardial infarct.
6. Myocardial infarct without obvious complete coronary occlusion.

Our material at the Massachusetts General Hospital is not of such a nature that we can report the refinements of examination by injection techniques. That is, we are not able to talk about what wasn't found in these hearts but what was obvious in routine examination of the entire heart by gross and microscopic methods.

The following cases illustrate the high degree of coronary pathology found at autopsy in certain patients with normal electrocardiograms in the weeks or months prior to death.

Female, Age 79

Present Illness—Phlebitis right leg. Vein ligated. No pulmonary infarcts. Died of pneumonia.
Heart symptoms—Dyspnea and orthopnea. Digitalis given.
Heart Examination—Negative.
Autopsy—Heart, 300 gms. Patchy, pale, gray, partial fibrosis of myocardium of left ventricle in posterior aspect near the A-V groove. Scarring of papillary muscles of mitral valve. Coronaries—focally arteriosclerotic and partially calcified. Lumina markedly reduced in size, and in one or two areas recanalized. Partial occlusion with recanalization—right coronary (2 points), and in one point after crossing over to left ventricle; left coronary 1 cm. from the bifurcation, stony hard, calcified, and partially occluded by atheroma for 1 cm.
Microscopic—Heart, myocardium—markedly scarred. Small collections of lymphocytes in fibrous patches. Similar changes in papillary muscles.
Electrocardiogram—November 3, 1939—normal rhythm, rate 90, rather small complexes and rather low T waves. Normal amplitude chest lead. (Tracing taken forty-two days before death.)

Male, Age 73

Present Illness—Post-operative prostatectomy. Acute retention.
Heart Symptoms—None
Heart Examination—Not enlarged. Sounds faint. No murmurs. Premature beats. Pipe-like peripheral vessels. Blood pressure 115/80.
Diagnosis—Perforated urethra with extravasation of urine.
Autopsy—Heart weight 300 gms. There was marked thinning of the posterior lateral portion of the right ventricle and replacement by fibrous tissue over an area 3 cm. in diameter. Coronaries—complete occlusion of right descending branch 5 cm. from its origin. Well organized thrombus with complete adherence to the wall. Marked sclerosis of other vessels but no narrowing.
Microscopic—Marked fatty infiltration between the muscle bundles of the right ventricle.
Electrocardiogram—December 29, 1939—Auricular premature beats, slightly slurred QRS complexes, and slight left axis deviation. (Tracing taken one week before death.)

Male, age 70

Present Illness—19 months of urinary obstruction from enlarged prostate.
Heart Symptoms—None.
Heart Examination—Blood pressure 150/70. Heart not enlarged. Regular rhythm except for premature beats.
Diagnosis—Heart weight 350 gms. Normal muscle. Endocardium, left ventricle grayish and very slightly thickened. **Coronaries: very high degree of arteriosclerosis with calcification** but wide lumen throughout.
Microscopic—Heart muscle negative.
Electrocardiogram—November 18, 1939—normal rhythm, rate 75, slight left axis deviation. (This tracing was taken two months and nine days before death.)

Male, Age 68

Present Illness—Rectal bleeding—2 months.
Heart Symptoms—None
Heart Examination—Blood pressure 170/82. Heart sounds distant. Soft systolic murmur at the left sternal border. Left ventricular hypertrophy by x-ray.
Diagnosis—Cancer of the rectum. Operated. Hypertensive heart.
Autopsy—Heart enlarged, 500 gms. Moderate calcification of aortic and mitral valves, numerous discrete and confluent yellow atheromatous plaques with calcification and definite narrowing of lumen of smaller branches. No infarcts.
Microscopic—Heart, muscle fibers showed some degree of hypertrophy.
Electrocardiogram—February 20, 1940—auricular and ventricular premature beats with left axis deviation, and slightly inverted T three. (Tracing taken nine days before death.)

Male, Age 63

Present Illness—(Healed tuberculosis) Hypertension. Paroxysmal auricular fibrillation—April, 1939. Congestive failure with paroxysmal tachycardia—November, 1939, and return of auricular fibrillation with death from congestive failure. Chronic nephritis. Uremia.
Heart Examination—Heart a little enlarged. Good heart sounds. Aortic second sound plus. Slight aortic systolic murmur.
Diagnosis—Hypertensive heart disease.
Autopsy—Chronic vascular nephritis, hypertensive heart, marked coronary sclerosis. Agenesis right kidney. Old coronary thrombosis. Heart weight—710 gms. Firm, pale myocardium. Markedly atheromatous mitral valve. All major branches of **coronary arteries markedly thickened, and hardened, cutting** with extreme difficulty because of calcium. Atheroma narrows most of them to ⅓ to ¼ normal diameter. In circumflex branch of left, there is a focus of thrombosis 5 mm. in length. Section shows minute canalizing vessel at this point.
Electrocardiogram—November 3, 1939—normal rhythm, rate 90, slight left axis deviation. (Tracing taken two months and nine days before death.)

These examples are not shown to discredit the electrocardiograph. They are shown in the interest of a vigilant skepticism. There is no question but what an electrocardiogram is invaluable in acute coronary insufficiency, and in acute myocardial infarction, and it may be in the healed stage with myocardial scarring. It is wise to remember, however, that enough instances exist in which gross coronary pathology is present with normal electrocardiograms to indicate that humility should be a virtue of electrocardiographers. An electrocardiogram never tells what a heart may be able to perform, and frequently reveals little or nothing of its structural defects. If it is believed, that electrocardiographic changes in any case are to be interpreted as indicating coronary atherosclerosis, we should be prepared to say that they mean either acute coronary ischemia, or some degree of myocardial infarction, and we should not rely on a diagnosis of "coronary sclerosis", or "coronary narrowing" from the electrocardiogram alone. The over-diagnosing of "myocardial degeneration", or of "coronary disease" on the basis of equivocal electrocardiograms often results in disastrous alterations of patients' lives and happiness.

On the other hand, normal electrocardiograms may be found, not only with high degrees of coronary sclerosis and narrowing, but also in some cases with definite myocardial fibrosis or infarct.

It is not generally recognized that the superimposing of a basal infarct upon an old anterior infarct may actually raise the T wave in lead one and apparently improve the electrocardiogram, when the myocardium is actually in a more damaged state.

A diagnosis of the type or degree of heart disease from which a patient is suffering should not be made from the electrocardiogram without knowledge of the clinical condition of the patient at the time of the recording of the tracing.

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